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Stress Response and Emotional Security in the Intergeneration Transmission of Depressive Symptoms

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STRESS RESPONSE AND EMOTIONAL SECURITY IN
THE INTERGENERATIONAL TRANSMISSION
OF DEPRESSIVE SYMPTOMS

By

Kristen Wilkinson

An Honors Project Submitted in Partial Fulfillment

of the Requirements for Honors

in

The Department of Psychology

The School of Arts and Sciences

Rhode Island College

2014

STRESS RESPONSE AND EMOTIONAL SECURITY IN THE INTERGENERATIONAL TRANSMISSION OF
DEPRESSIVE SYMPTOMS

An Undergraduate Honors Project Presented

By

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To

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Abstract

Few studies have examined possible explanations (i.e., examining mediators) as to why depressive symptoms are transmitted from mothers to adolescents, as well as neglected to consider which adolescents are most vulnerable to this transmission (i.e., examining moderators). Thus, the aim of this study is to focus on stress reactivity as a moderator of the transmission of depression from mothers to adolescents through emotional insecurity. Ninety-three mother-adolescent dyads were examined, with adolescents between the ages of 13 to 17. Data was collected in the home through surveys, a mother-adolescent interaction task and physiological measures from the adolescent to examine stress response. Results suggested that emotional insecurity mediated the relationship between maternal and adolescent depressive symptoms. Findings also provided some support that higher baseline stress response acted as a partial moderator such that adolescents who evidenced a higher stress response at baseline appeared more vulnerable to the transmission of depression.

Stress Response and Emotional Security in the Intergenerational Transmission of Depressive Symptoms

Depression is a growing area of concern in America. Depressive disorders are accountable for several deaths a year and according to the National Institute of Mental Health, about seven in every 100,000 adolescents commit suicide in America every year due to depression (Insel, 2012). Depression may be intergenerational as studies indicate that a correlation exists between maternal depression and adolescent depression (Hammen, Brennan & Brocque, 2011; Nelson, Hammen, Brennan & Ullman, 2003). Mothers who exhibit depressive symptoms often do not have a warm and secure relationship with their adolescents, placing adolescents at risk for developing depressive symptoms (Teti, Gelfand, Messinger & Isabella, 2011). Thus, one reason that depressive symptoms may be passed down from mothers to their adolescents is because maternal depressive symptoms impact adolescents' sense of emotional security within the parent-adolescent relationship, which in turn may lead to increased depressive symptoms among adolescents (Forman & Davies, 2003; Milan, Snow & Belay, 2009). To date, however, few studies have examined mechanisms that may help explain the transmission of depression from mothers to children during adolescence, with most studies only examining this transmission through emotional security in childhood (Milan et al., 2009).

In addition to identifying possible mechanisms that explain the transmission of depressive symptoms from mother to adolescent, it is also important to examine which adolescents might be most vulnerable to this transmission. Stress has an important impact on the body and how emotions are managed and may act as a vulnerability for this transmission of depression (Ellis & Boyce, 2008). Specifically, if a child evidences a dysregulated stress

response, specifically heightened reactivity, they will be particularly vulnerable to this transmission of depression because they are highly sensitive to context (e.g., maternal depression). This vulnerability may result in adolescents having a greater risk of developing emotional insecurity and in turn, reporting more depressive symptoms. To our knowledge, research has not examined how adolescents' stress response may increase the transmission of depression symptoms from mother to adolescent. Thus, the goal of this study is to examine if adolescents' stress response, as measured by heart rate blood pressure, and cortisol moderates the effects of maternal depressive symptoms and emotional security on adolescents' depressive symptoms.

Transmission of Depressive Symptoms

Maternal and Adolescent Depressive Symptoms

There is research to support that maternal depression and adolescent depression are highly correlated with each other, suggesting that depressive symptoms are passed down in families and children of depressed mothers are at an increased risk of developing depression later in life (Hammen et al., 2011; Milan et al., 2009). According to DSM-V criteria, depressive symptoms can include depressed mood/affect or a loss of interest in activities for at least a two week period (American Psychiatric Association, 2013). Munoz, Beardslee and Leykin (2012) explained that depression can be caused by specific and nonspecific factors. A specific factor can be an immediate family member who has depression (i.e. mother) and a nonspecific factor can be the childhood that was experienced (e.g., a negative environment of poor parenting). If an adolescent has both of these factors, they are at a high risk for depression.

Hammen and colleagues (2011) described the transmission of depression between mothers and their children as intergenerational and a vicious cycle. Results from their study

suggested that depressed mothers who had depressed parents were at high risk for parenting characterized by low levels of maternal warmth and high levels of hostility and inconsistency. This study found that negative parenting resulted in more depression in children who would grow up and eventually use the same negative parenting strategies when the depressed children had children of their own. The continuing cycle of transmitting depression from generation to generation through non-specific factors such as parenting was typical in many families in this study and was centered around parenting and adolescents' sense of emotional security in the parent-adolescent relationship.

Transmission of Depressive Symptoms through Emotional Security

There are several different factors that may link maternal depressive symptoms to depressive symptoms in adolescents. Milan and colleagues (2009) conducted an eight year longitudinal study of three-year old children and found that the transmission of depression is influenced by a mediating factor, adolescent's sense of security, as well as the severity of depression in the maternal caregiver. Emotional security of the child was a prevalent factor in this study, a construct that stems from attachment theory. In attachment theory, children can either have a sense of felt security or insecurity depending on the family context and parenting behaviors. Secure children typically have a warm relationship with their mothers and are able to regulate their emotions. However, insecure children do not seek help from their mother and are unable to regulate their emotions (Milan et al., 2009).

Davies and Cummings (1994) introduced an extension to attachment theory called emotional security theory. A child's sense of emotional security is based on past experiences between the parent and child that the child uses to cope with certain situations in the present and the future. Emotional security is the driving force behind emotion regulation and coping in the

face of stress. If a child is introduced to a destructive environment while growing up, the child would be less likely to develop a secure sense of emotional security for future experiences in life. For example, if a child is around marital conflict at a young age with no resolution between the parents and if the child does not receive positive affect from either parent, the youth will experience problems feeling emotionally secure within the home environment, which may result in negative affect and depressive symptoms.

Forman and Davies (2003) suggested that adolescence may be a particularly critical period for developing a sense of security in the family context and may put adolescents at risk for psychological problems (e.g., depressive symptoms). In a longitudinal study with 220 young adolescents, these researchers found that instability within the family (e.g. lack of warmth from the maternal caregiver) was found to have a negative effect on the adolescent's sense of emotional security and put the adolescent at risk for psychological problems later in life. In a related study by Forehand, Brody, Slotkin, Fauber, McCombs and Long (1988), sixty-nine adolescents and their mothers were assessed using self-report and two interaction tasks (spread out across a year). Results supported the contention that children who have a lower sense of parental warmth and who did not have a secure relationship with their mothers were more at risk for depression. These studies suggested that emotional security may play an important role in adolescents' depressive symptoms.

Few studies have examined the pathway between maternal depressive symptoms and adolescent depressive symptoms through emotional security. Hammen and colleagues (2011) conducted a study that focused on the intergenerational transmission of depression and childrearing. That study found that depressed mothers who had children at a younger age were at a higher risk for a negative parenting style, which has an impact on the child's sense of

emotional security. This unhealthy family environment was associated with a greater risk for depression symptoms in their child because the child did not have an emotionally secure relationship with their depressed mother. This study highlighted the importance of the quality of mother-child relationships, an element linked to emotional security, as a predictor of the transmission of depressive symptoms.

Teti and colleagues (1995) also examined the transmission of depressive symptoms from mothers to infant children. Results indicated that children who had strategies for coping with stressful situations among their attachment figure (secure or insecure) were less at risk for depressive symptoms than children with no attachment strategies (disorganized attachment). Although some children had an insecure attachment with their attachment figures, they were able to form logical strategies; however, these children were still at high risk for depressive symptoms. Further results of this study showed that about 80% of children with depressed mothers had an insecure attachment. The small percentage of children with a secure attachment to their mothers were less stressed (through observational analysis) and expressed low levels of depressive symptoms, suggesting that children's handling of stress may make them more or less vulnerable to the transmission of depressive symptoms. The current study will build on this research and examine the full pathway from maternal depressive symptoms to adolescent depressive symptoms through the mediator of emotional insecurity.

Stress Response

Adolescents' stress response may increase adolescents' vulnerability for the intergenerational transmission of depressive symptoms. When an adolescent is exposed to adversity over a long period of time, there are two ways in which adolescents' bodies might respond in a way that is indicative of a dysregulated stress response, used by the adolescent to

cope with the adversity. The first dysregulated stress response is blunted or low stress reactivity (hypoarousal), where the adolescent shows very low stress levels during stressful situations. The second dysregulated stress response, which is the focus of this study, is heightened stress reactivity (hyperarousal), where adolescents show very high levels of stress during and after stressful situations (Davies, Sturge-Apple, Cicchetti & Cummings, 2007). Stress response can be measured through heart rate, blood pressure and through salivary cortisol. When the body experiences a stressful situation, the necessary systems are inhibited while others remain activated. This process is vital in the short term, however, if this response remains active over long periods of time, it can lead to a dysregulation of the body's natural stress response both in times of acute stress and times of rest.

Ellis and Boyce (2008) proposed the biological sensitivity to context theory (BSC), which suggested that adolescents interpret stressful situations depending on their level of stress reactivity, and the context in which they developed. These individual differences in reactivity may have developed as a result of the interaction between genetic and early environmental experiences. This concept is similar to the diathesis-stress perspective in which an individual has a vulnerability to develop a dysregulated stress response due to genetic or biological factors (Ingram & Luxton, 2005). This vulnerability increases adolescents' chances of developing depression if they are exposed to stressful life experiences. Ellis and Boyce (2008) found that adolescents who have high stress reactivity are very sensitive to context, meaning that adolescents with this stress response are sensitive to the effects of both positive and negative environments on development. If an adolescent was exposed to a stressful environment, such as the environment that may be experienced in homes where mothers report depressive symptoms, the adolescent will be at a greater risk for health or psychological problems later on in

development. However, if the adolescent was brought up in a low stress, supportive environment the adolescent will be more likely to be successful and flourish. The BSC theory explains that high stress reactivity is not necessarily a vulnerability, however, it can create a high vulnerability if the surrounding environment is negative (e.g. family adversity, maternal depression). This view differs slightly from that of a diathesis-stress perspective, which would view adolescents' stress response as only a vulnerability.

Research supports the BSC theory; Obradovic, Bush, Stamperdahl, Adler and Boyce (2010) found that a heightened stress response yielded positive or negative outcomes depending on the context. These researchers' suggested that children with a heightened stress response were at an increased risk for behavioral and health problems in high stress environments (e.g. marital conflict or divorce). The researchers also found that children who had a heightened stress response and were in low stress environments did not show risk for behavioral or health problems. In a laboratory study, Obradovic, Bush and Boyce (2011) elicited stress response from children using different challenging tasks. The researchers found that highly reactive children were highly vulnerable to adversity and low quality environments. Results supported that high stress reactivity was more of a process that developed throughout early childhood depending on the environment and how frequent the child was faced with adversity or lack of parental engagement. Highly reactive children that were faced with a difficult task or marital conflict displayed higher levels of anxiety, frustration and other internalizing symptoms because highly reactive children were sensitive to context and had a difficult time managing negative emotions.

Emotional Security, Stress Response and Depression

Adolescents with heightened stress reactivity and an emotionally insecure relationship with their depressed mothers may be more vulnerable to developing depressive symptoms.

Studies have not specifically examined the impact that stress reactivity may have on the transmission of depressive symptoms from mothers to adolescents. However, related research by Lupien, McEwen, Gunnar and Heim et al. (2009) examined the effects of stress on both animals and humans. These researchers found that parent-child interactions as well as the psychological state of the mother had an effect on a child's stress and behaviors in the future. If a child does not receive adequate care and attention from their mother, they are vulnerable for health problems especially if the child is introduced to stressful situations. According to Lupien et al. (2009), adolescence is the point in which the brain is most vulnerable to stress. If an adolescent does not experience a warm secure relationship with their maternal caregiver, the adolescent will be vulnerable to stressful environments because they will not have the experience to handle and cope with stress. As a result, the adolescent will be at risk for developing psychopathology.

Furthermore, a recent study by Waugh, Muhtadie, Thompson, Joormann and Gotlib (2012) examined how stress affected young girls that were at risk for depression. The researchers found that dysregulation of stress in young girls of depressed mothers put the girls at risk for depressive symptoms. Girls that had a dysregulated stress response and received negative affect from their mothers displayed higher depressive symptoms. This research provides preliminary evidence that the transmission of maternal depressive symptoms to adolescents through emotional insecurity is exasperated for adolescents who evidence a heightened stress response.

Hypotheses

There are several studies that support the relationship between adolescent depression and maternal depression (Hammen et al., 2011; Milan et al., 2009; Munoz et al., 2012). These studies are limited, however, because they do not focus on emotional security as a possible explanation as to why depressive symptoms may be transmitted from mothers to adolescents and they do not

look at possible moderators (e.g. heightened stress reactivity) to this entire pathway. Thus, this study examines whether a dysregulated stress response in adolescents of mothers with higher depressive symptoms creates a vulnerability for depressive symptoms in their adolescents. Specifically, this study has two hypotheses. We hypothesize that emotional security will act as a mediator between the transmission of depressive symptoms such that one reason why maternal depressive symptoms and adolescent depressive symptoms will be associated is because adolescents develop a sense of emotional insecurity. Secondly, we hypothesize that a dysregulated stress response will heighten the negative impact of maternal depressive symptoms on emotional insecurity and adolescent depressive symptoms, such that adolescents with a heightened stress response will be more at risk for the negative effects of maternal depression than adolescents who do not evidence a dysregulated stress response (Figure 1). These findings will contribute to the limited research on stress reactivity as a moderator of emotional insecurity in the parent-adolescent relationship and allow further research to be done on this topic. Results from this study may assist programs for adolescents and their mother's with depression by giving more information on ways to avoid/help depression and the potential sources of depressive symptoms in adolescents.

Insert Figure 1

Method

Participants

Participants were 93 adolescents (70% female, 77% Caucasian) and their maternal caregivers (91% biological) who live within a 15 mile radius of the Providence County in Rhode

Island. Adolescents in 9th thru 11th grade between the ages of 13 and 17 were selected ($M=15.1$, $SD = 0.90$). Exclusion criteria included (a) families making less than \$40,000 a year, (b) adolescents with an IQ less than 70, and (c) primary spoken language not English. Participants were required to speak English as their primary language in order to be included in the study given there were no researchers in the study that were fluent in another language. The sample was a convenience sample. Recruitment methods included flyers posted in the local area, businesses and places that parents and adolescents frequent (e.g., library, coffee shops), the American Student List (commercial mailing list) and speaking to adolescents and parents at local events, as well as handing out information at some private schools in the area.

Procedure

Data were collected through home visits by graduate and undergraduate research assistants. Prior to the home visit, consent was obtained from the mothers, fathers and the adolescents. The home visit was approximately two and a half hours long. During that time, measures of heart rate, blood pressure and cortisol were obtained from the adolescent at five different time periods (baseline, right after stressor, and the recovery period after the stressor) in order to measure how the adolescent copes with stressful situations. To obtain the baseline, the adolescent watched a relaxation DVD (Sponge bob or a nature video) for 20 minutes and measures of heart rate, blood pressure and cortisol were obtained twice during that time (at the halfway point and at the conclusion of the video).

Surveys were also given to both the adolescent and the mother to measure demographics, parenting and internalizing/externalizing behaviors. Two videotaped interaction tasks using the Autonomy and Relatedness Coding System (Allen, Hauser, Eickholt, Bell, & O'Connor, 1994) also took place between the mother and the adolescent about an issue of conflict within the home

and outside of the home. The issue of conflict within the home was obtained through a Family Issues Checklist (Robin & Foster, 1989), which included topics of conflict such as doing homework and coming home on time. Response options were between one (*calm*) and five (*angry*), the overall highest rated conflict between the mother and the adolescent was chosen. The issue of conflict outside of the home was obtained by asking the adolescent to identify an issue (e.g. a problem with a friend) that they are experiencing outside of the home. Each interaction task was eight minutes long and heart rate was obtained every 30 seconds throughout each interaction task. The purpose of the interaction tasks for this study was to induce stress that adolescents might experience in everyday interactions with their parent. The mother and adolescent were compensated \$40 each for their time on the study.

Three consent documents were sent to the mother and adolescent; one consent document for the mother to participate, one assent document for the adolescent to participate, and a consent document to be signed by both the mother and the adolescent's father or other legal guardian giving permission for the adolescent to participate in the project. Referrals to counselors were given to all families with the consent forms in case of any distress experienced throughout the study. Potential ethical concerns were addressed. For example, the family is prompted that they do not need to continue the interaction and stop at any time if they feel uncomfortable or upset when discussing the issue of conflict. Adolescents are also prompted by the research assistants at the beginning of the survey and throughout the visit that all information is strictly confidential and the adolescent can stop at any time if they feel uncomfortable at any point. All participant names, addresses, and other information are not be linked to the research data (ID numbers are given to each family to maintain this confidentiality).

Measures

Control variables. Gender was the only control included in these analyses because it was the only variable that had a significant relationship with other variables of interest in this study. Male participants were coded as a zero and female participants were coded as a one.

Maternal depressive symptoms. The Center for Epidemiologic Studies Depression Scale (CES-D Scale; Radloff 1977) was used to measure Maternal Depressive symptoms. Depressive behaviors on the CES-D Scale include statements such as “I felt everything I did was an effort” and “I felt lonely.” Response options range from a 1 (*Rarely or none of the time*) to a 4 (*Most or all of the time*) where higher scores represented a higher level of depressive symptoms. The CES-D is a widely used instrument that has been found to have good reliability ($\alpha=.89$) as demonstrated by internal consistency and has been shown to be correlated with other measures of depressive symptoms (Radloff, 1977).

Parent/Adolescent emotional security. Emotional security was measured through the Inventory of Parent and Peer Attachment (IPPA; Armsden & Greenberg, 1987) on the adolescent survey which assesses warmth, support, communication, and alienation in the parent-adolescent relationship. Sample items include “My mother accepts me as I am” and “When we discuss things, my mother cares about my point of view.” Responses range from a 1 (*Almost Always or Always True*) to a 5 (*Almost Never or Never True*) where higher scores represent a lower level of emotional security. This scale has been used in numerous studies to describe attachment and the parent-adolescent relationship (El-Sheikh & Elmore-Staton, 2004). The IPPA had high reliability ($\alpha=.94$) and past research has demonstrated convergent validity in regards to parent attachment through trust and communication scores (Armsden & Greenberg, 1987).

Stress response. Stress response was measured through obtained salivary samples from

the adolescent in order to obtain cortisol, which is a naturally occurring hormone in the body that can measure stress. Samples were obtained non-invasively using a cotton strip that the adolescent was asked to stick between their back molars and the inside of their cheek for approximately two minutes. Upon completion, the cotton strip was placed in a tube and then directly on ice stored at -40C. The samples were taken during a relaxation period before the interaction task in order to obtain a baseline. Samples were taken again after the parent-adolescent interaction period to measure the adolescent's stress response. Three samples were taken at 15 minute intervals throughout the rest of the home visit to not only measure a peak in cortisol (15-30 minutes after task) but also a recovery in cortisol.

Stress response was also measured through cardiovascular response. A Contec 08C blood pressure cuff was used to assess systolic blood pressure (SBP) and diastolic blood pressure (DBP). A Pulsox sensor was attached to the adolescent's finger to assess heart rate (HR). Blood pressure and heart rate was obtained during each salivary sample time point and in the middle of the two interaction tasks. Heart rate was obtained every 30 seconds during each interaction task.

For purposes of this study we measured stress response by utilizing baseline levels of stress response variables at the beginning of the visit as opposed to reactivity to the stressor as adolescents did not evidence a predictable pattern of stress reactivity to the interaction task for all stress response variables.

Adolescent depressive symptoms. Adolescent depressive symptoms were measured from the Child Behavior Checklist Scale (YSR; Achenbach, 1991a & CBCL; Achenbach, 1991b) from both the mother's and the adolescent's survey. Statements include "*I feel confused or in a fog*" and "*I am overtired.*" Responses range from 1 (*Not true*) to 3 (*Very/Often true*) where higher scores represent a higher level of internalizing behaviors. The CBCL and YSR are found

to have high internal construct validity and displayed high reliability ($\alpha=.85, .90$; respectively) in identifying internalizing and externalizing behaviors in adolescents (Achenbach, 1991a & Achenbach, 1991b). For purposes of this project, adolescent's report on the YSR and the mother's report on the CBCL were used to create an average variable for adolescent depressive symptoms.

Analytic Strategy

Descriptive Analyses

We examined whether heightened stress reactivity created a vulnerability for depressive symptoms in emotionally insecure adolescents with depressed mothers. Path analysis was used in this study where the independent variables (Maternal depressive symptoms, and stress reactivity) were categorical and continuous and the dependent variables (emotional insecurity and adolescent depressive symptoms) were continuous. We examined three hypotheses (1) Maternal depressive symptoms are associated with increased adolescent depressive symptoms, (2) Emotional insecurity partially explains the intergenerational transmission of depression, and (3) Adolescents' stress response creates a vulnerability in which adolescents with emotionally insecure relationships with their mothers will be more at risk for the transmission of depression. Means and standard deviations were obtained for all variables (Table 1).

Inferential Analyses

The AMOS 20.0 structural equation-modeling program (SEM) was used to estimate path models. Model fit for path models was examined using the chi-square goodness of fit statistic, the comparative fit indices (CFI), and the root mean square error of approximation (RMSEA). A nonsignificant chi-square indicated a good model fit (Byrne, 2001). CFI values of .95 or higher indicated a good model fit and RMSEA values below .05 indicated a good model fit and below

.08 an adequate model fit (Thompson, 2000). Error variances were included for all the dependent variables to account for within group differences. The significance of all models was set at $p < .05$.

The default in the AMOS program for missing values is the full information maximum likelihood estimation procedure (FIML), which was used in this study. Winsoring was used for the stress reactivity measures that were three standard deviations away from the mean. Data is assumed to be normal in order to use parametric statistics, however, with physiological measures, data tends to be skewed due to the high variability in participants. Past literature supports the use of winsoring in order for data to be closer to the mean as opposed to large outliers (Duan, 1999).

The first step in data analyses examined the direct relationship between maternal depressive symptoms and adolescents depressive symptoms. The second step included the emotional insecurity variable in the model to see if it minimized the direct relationship between maternal depression and adolescent depression. Sobel's formula also was used in order to test the mediating pathway for statistical significance.

To examine our moderating hypothesis, we created a median split of the baseline heart rate, cortisol and both the systolic and diastolic blood pressures, where the top halves of participants were labeled higher reactivity with a score of one and every other participant received a score of zero. This method, as opposed to taking the top 75%, was used due to our small sample size. These variables were entered as a grouping variable in four separate multiple group models in AMOS. Multiple group models allow researchers to test if the regression pathways differ across groups. This was accomplished by comparing a model that is fully constrained to a model that allows the regression pathways to vary across the groups (e.g.,

maternal depression to emotional insecurity). When the multiple group model was fully constrained, the two groups are assumed to be equal across all parameters, including regression pathways, and when the model allows the pathways to vary the results display the pathways in which the two groups differ.

To examine if moderation was present we examined several criteria that included chi square difference tests, critical ratios, and if coefficients differed across the models in their significance level. In the context of multiple group models, chi square difference tests compare a model where pathways are assumed to be equal across groups and a model where pathways are assumed to be different across groups, with a significant p-value indicating that the model where paths are allowed to vary across groups is the correct model. Standardized coefficients and critical ratios were also examined for other evidence of moderation for each of the multiple group models (cortisol, heart rate, systolic and diastolic blood pressure). Critical ratios above 1.96 indicated a significant difference in the regression pathways across models.

Results

Mediation

For our mediating hypotheses, in the direct effect model we found a significant association between maternal depressive symptoms and adolescent depressive symptoms ($\beta = .20, p = .04$), indicating that higher maternal reports of depressive symptoms were correlated with higher reports of adolescent depressive symptoms. Gender was significantly associated with adolescent depressive symptoms ($\beta = .30, p = .01$). When emotional insecurity was included into the model, the direct effect between maternal and adolescent depressive symptoms was reduced to non-significance ($\beta = .15, p = .14$), indicating mediation. Maternal depressive symptoms were related to higher emotional insecurity ($\beta = .23, p = .03$) and higher emotional insecurity was

related to increased adolescent depressive symptoms ($\beta=.26, p=.01$). Gender was significantly related to adolescent depressive symptoms ($\beta=.27, p=.01$), such that being a female had a stronger relationship with depressive symptoms than being a male. Sobel's test was used to test for mediation and reached significance at $z = 2.40, p=.02$. Comparative fit index (CFI) = .96 indicated a good model fit and RMSEA = .08 indicated an adequate fit to the mediating model. The chi-square of the model was non-significant $\chi^2 = 1.72, df = 1, p > .05$.

Moderation

Heart rate. Results from the group difference test indicated that there was not a significant change in chi-square when the paths were allowed to differ for those high in stress response and those low in stress response, $\Delta\chi^2 = 4.03, df = 4, p > .05$. Critical ratios indicated that one of the structural pathways differed across stress response; the pathway from maternal depressive symptoms to emotional insecurity for higher baseline stress response individuals yielded a significant critical ratio indicating differences across groups (CR) = -1.96. Specifically, for participants who evidenced a higher heart rate at baseline, maternal depressive symptoms were correlated with emotional insecurity ($\beta=.26, p=.02$) but for participants who evidenced a lower stress response at baseline, maternal depressive symptoms were not correlated with emotional insecurity ($\beta=.15, p=.16$) indicating that having a higher stress response at baseline may act as a vulnerability that exacerbates the impact of maternal depression on emotional insecurity.

Diastolic blood pressure. Results from the group difference test indicated that there was not a significant change in chi-square when the paths were allowed to vary for those high in stress response and those low in stress response, $\Delta\chi^2 = 4.83, df = 4, p > .05$. None of the critical ratios reached 1.96. Although the omnibus test results did not meet requirements for moderation,

a closer examination of the betas across the two models indicated some evidence of moderation. Individuals with higher baseline stress response yielded significant findings from emotional insecurity to adolescent depressive symptoms ($\beta=.43, p=.00$) but for other individuals that evidenced a lower baseline stress response, the pathway from emotional insecurity to adolescent depressive symptoms was not significant ($\beta=.13, p=.33$).

Systolic blood pressure. Results from the group difference test indicated that there was a significant change in chi-square for those high in stress response and those low in stress response, $\Delta\chi^2 = 10.30, df = 4, p < .05$. Interestingly, none of the individual critical ratios were above the 1.96 threshold. However, the higher baseline stress response group yielded significant findings from emotional insecurity to adolescent depressive symptoms ($\beta=.40, p=.00$) but emotional insecurity to adolescent depressive symptoms was not significant ($\beta=.18, p=.17$) in the group evidencing a lower stress response at baseline.

Cortisol. Results from the group difference test indicated that there was not a significant change in chi-square when the paths were allowed to differ for those low and high in baseline cortisol levels, $\Delta\chi^2 = 1.04, df = 4, p > .05$. Critical ratios and regression coefficients across groups also did not differ.

Discussion

Findings from this study support that maternal depressive symptoms are correlated with adolescent depressive symptoms. Additionally, results support our mediating hypothesis where higher reports of emotional insecurity in the maternal-adolescent relationship may explain the transmission of depressive symptoms from mother to adolescent. These findings are consistent with past research suggesting that adolescence is a critical period for developing a sense of emotional security (Forman & Davies, 2003). When an adolescent is raised in an insecure

environment, the adolescent is at risk for developing psychopathology such as depression. These results also support past research that mothers who are depressed are more likely to display a lack of warmth with their adolescent, which can contribute to an adolescent's feeling of emotional insecurity (Hammen, Brennan & Brocque, 2011). In this context, the adolescent lacks the necessary experience and emotional regulation to handle stressful situations, which can be associated with depressive symptoms in adolescents.

Findings from our study also partially support that higher baseline adolescent stress response acts as a moderator for some physiological measures and may exacerbate the mediating pathway in emotionally insecure teens. Multiple group analyses found that individuals with higher baseline systolic and diastolic blood pressures were more vulnerable for the transmission of depressive symptoms through emotional insecurity than individuals with lower diastolic and systolic blood pressures. These findings support the diathesis-stress model (Ingram & Luxton, 2005) suggesting that individuals have a predisposed vulnerability to stress from early environmental and genetic factors that exasperates the impact of context on development. Individuals that are raised in an environment that is stressful and emotionally insecure may also have an innate trait vulnerability to stressful situations that may exacerbate these feelings of emotional insecurity. Unfortunately, we could not fully evaluate the theory of biological sensitivity to context because we did not examine if stress reactivity in youth may be helpful in more positive contexts.

Results provided some support that heart rate, diastolic and systolic blood pressures acted as potential vulnerabilities, however, cortisol at baseline did not act as a vulnerability for adolescent depressive symptoms. Past research has suggested that there is variability in the effect of stress response on adolescent outcomes, such that a blunted stress response may be associated

with psychopathology. For youth in our sample it is plausible that a blunted cortisol response may have acted as a vulnerability. Unfortunately, given the small sample size we were not able to examine multiple groups of stress response (hypo vs. hyper) in our analyses.

Gender was significantly associated with adolescents' depressive symptoms and this was more pronounced among youth that had a higher baseline heart rate, which has been supported in Waugh and colleagues' (2012) study that examined the effects of a dysregulated stress response (although we examined baseline data) among girls. This study found that girls with a higher stress response that received negative affect from their mothers were more at risk for developing depressive symptoms, indicating that dysregulation or a higher stress response acts as a vulnerability in the transmission of depressive symptoms, particularly for girls, which was also found in our study.

This study is not without limitations. The sample size was small given the use of multiple group path models and lacked diversity. Specifically, the sample was predominately female and upper-middle class. In future studies, larger populations should be looked at longitudinally with a more diverse sample. Furthermore, it is important to note that the data collected from this study is correlational, so the direction of effects cannot be determined. Limitations with the physiological data also should be noted. Physiological measures were not correlated with each other (Table 1) which may be due to delayed responses in some measures (heart rate vs. cortisol). Lack of correlations between physiological measures may also be due to different systems that each measure targets. For example, cortisol impacts the hypothalamic-pituitary-adrenal axis (HPA-axis) and heart rate/blood pressure impacts the sympathetic nervous system. Blood pressure was also separated as diastolic and systolic and it is possible that different participants formed the groups for each of our multiple group models. Future studies may want to examine

stress response as a holistic variable instead of four different variables.

Future studies can also examine the effects of parental psychopathology and marital conflict as contributors to the transmission of depression. These implications can be important in understanding the transmission of depressive symptoms in mothers and adolescents and can be used to set up future programs in the community that target adolescents experiencing depressive symptoms, where the quality of attachment and security as well as how the adolescent copes with stressful situations between the adolescent and their maternal caregivers will be the focus point.

Table 1.

Means, Standard Deviations and Correlations between Study Variables. (N= 93)

VARIABLES	1	2	3	4	5	6	7	8	9
1. Maternal Depression	_____								
2. Adolescent Depression-adolescent report	.13	_____							
3. Adolescent Depression-mom report	.34**	.58**	_____						
4. Emotional Insecurity	.23*	.36**	.23*	_____					
5. Gender	.17	.28**	.33**	.17	_____				
6. Heart Rate Baseline	.03	.10	.31**	.16	.25*	_____			
7. Systolic Blood Pressure Baseline	.03	.12	.10	.01	-.13	.05	_____		
8. Diastolic Blood Pressure Baseline	.11	.27*	.29**	.06	.25*	.26*	.58**	_____	
9. Cortisol Baseline	.04	-.18	-.00	-.02	-.17	-.13	-.04	.14	_____
M	1.50	1.49	1.38	2.16	N/A	76.48	112.50	65.15	8.26
SD	.43	.41	.31	.69	N/A	13.41	11.26	9.19	3.65

Note: * indicates a significant relationship at $p < .05$ and ** indicates a significant relationship at $p < .01$.

For gender, males coded zero and females were coded one.

Figure 1. Hypothesized Model

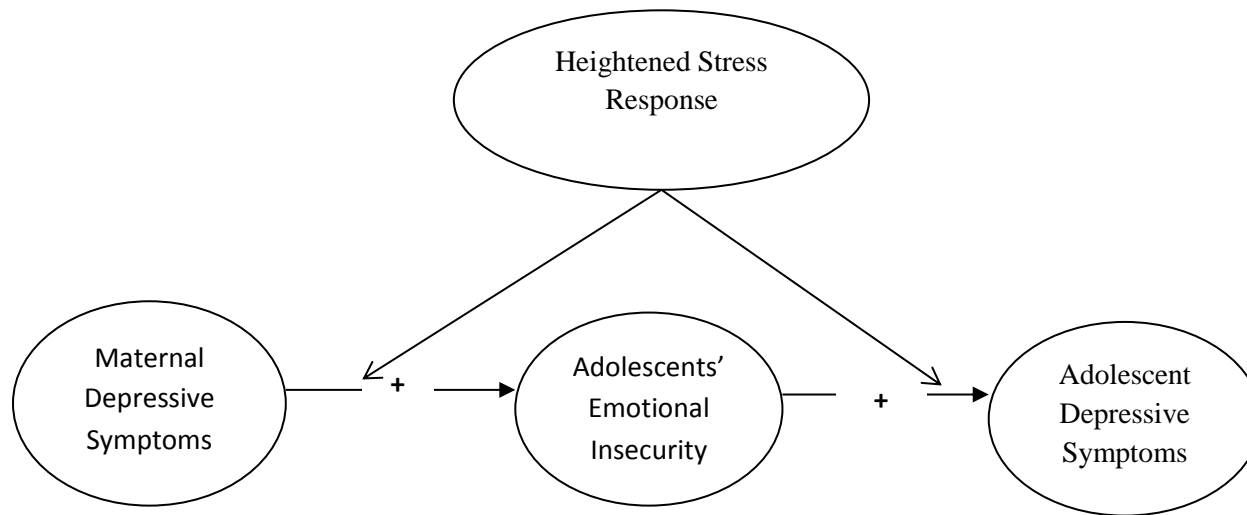


Figure 1. The Role of Stress Response in the Transmission of Depressive Symptoms through Emotional Insecurity. Note. Although not represented in this figure, we expect a direct relationship to exist between maternal depressive symptoms and adolescent depressive symptoms.

Figure 2. Mediating Model

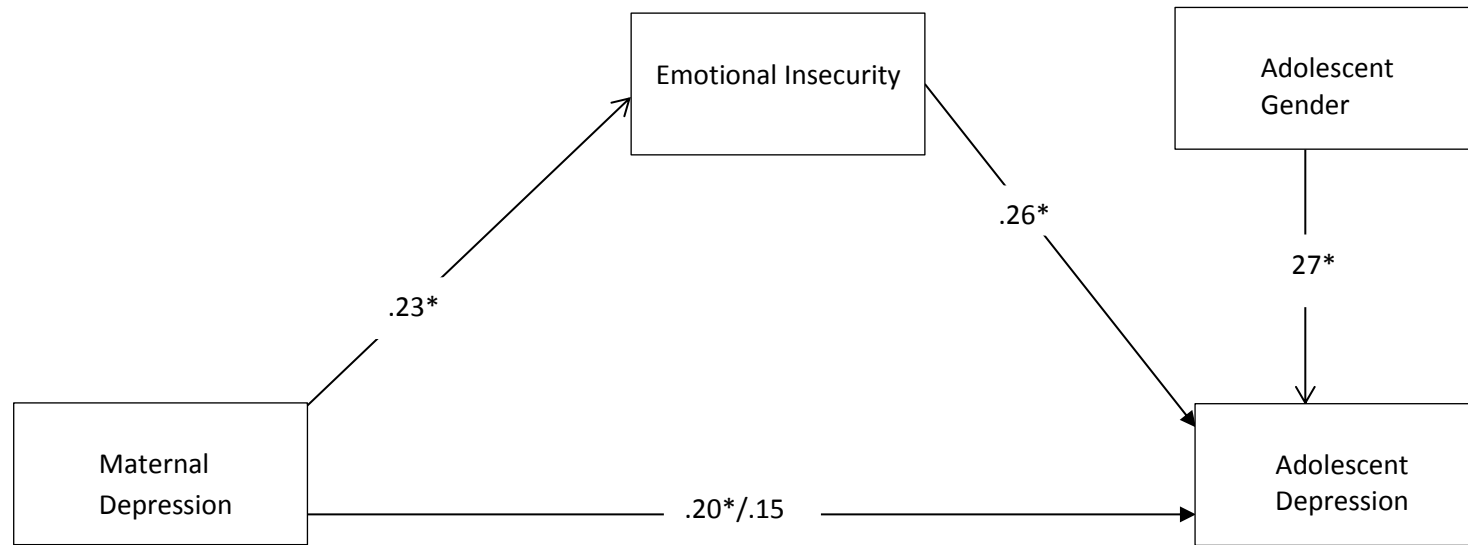


Figure 2. This model demonstrates the mediating effect of emotional insecurity on the relationship between mom and adolescent depressive symptoms. Partial mediation was found such that the main effect was reduced to non-significance when emotional insecurity was added to the model. (as indicated by the coefficients listed respectively). Error variances on dependent variables are estimated but not represented here. * indicates significant pathways at $p < .05$. $N = 93$ participants

Figure 3. Heart Rate as a Moderator

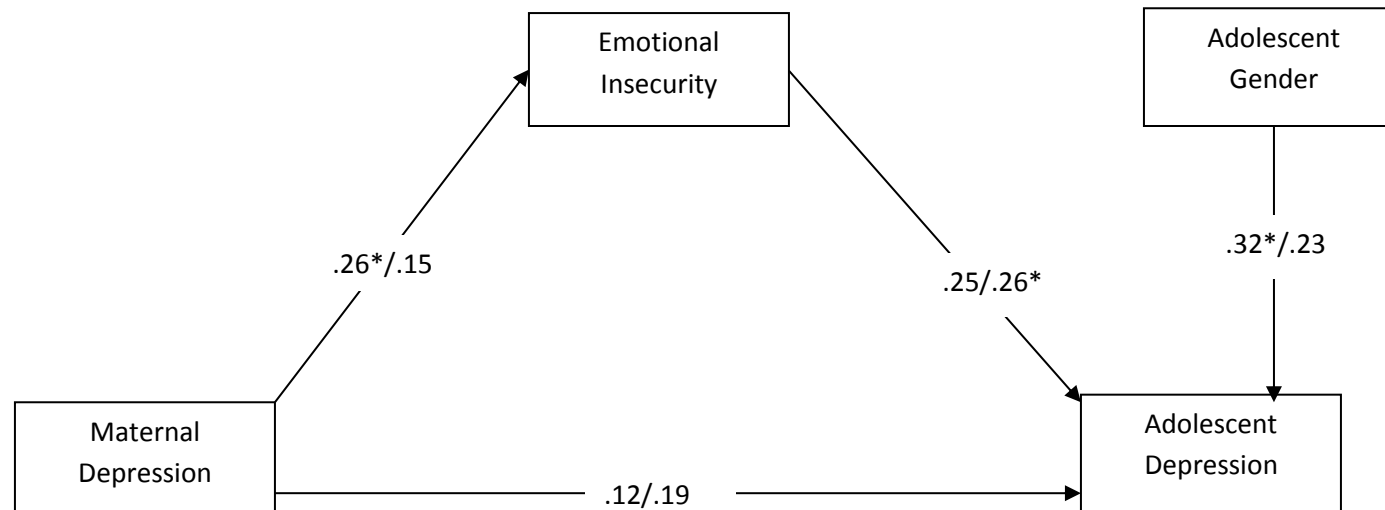


Figure 3. This model demonstrates the multiple group analysis of individuals with higher baseline heart rate (represented by first number) and individuals with lower baseline heart rate (represented by second number) when groups are allowed to differ. Error variances on dependent variables are estimated but not represented here. * indicates pathway is significant at $p < .05$. $N = 93$ participants

Figure 4. Diastolic Blood Pressure as a Moderator

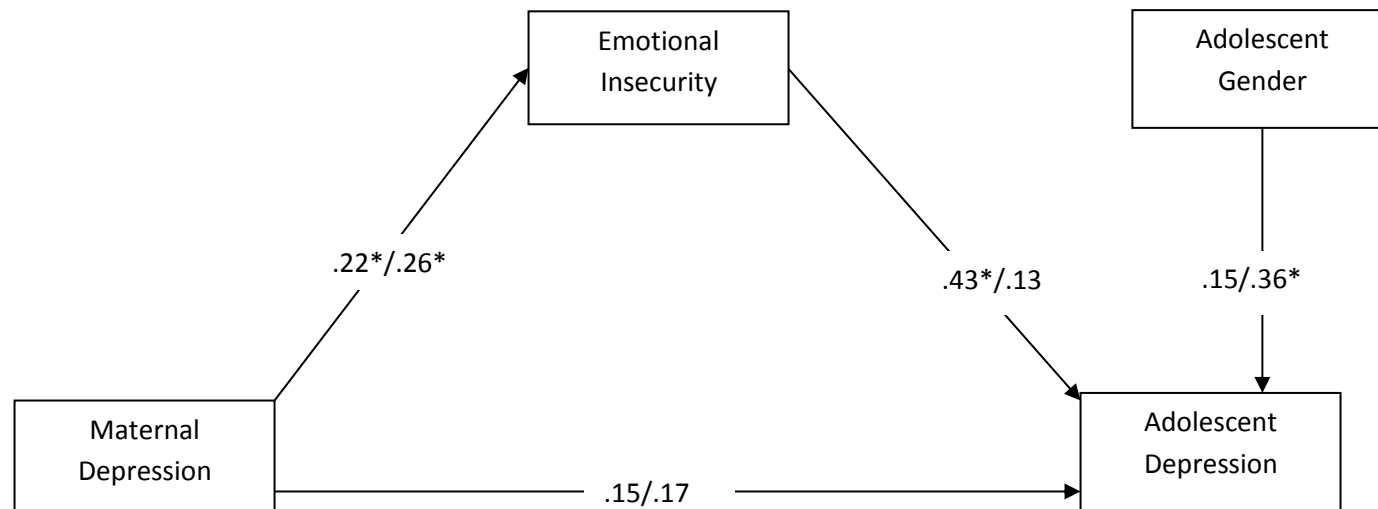


Figure 4. This model demonstrates the multiple group analysis of individuals with higher baseline diastolic blood pressure (represented by first number) and individuals with lower baseline diastolic blood pressure (represented by second number) when groups are allowed to differ. Error variances on dependent variables are estimated but not represented here. * indicates pathway is significant at $p < .05$. $N = 93$ participants

Figure 5. Systolic Blood Pressure as a Moderator

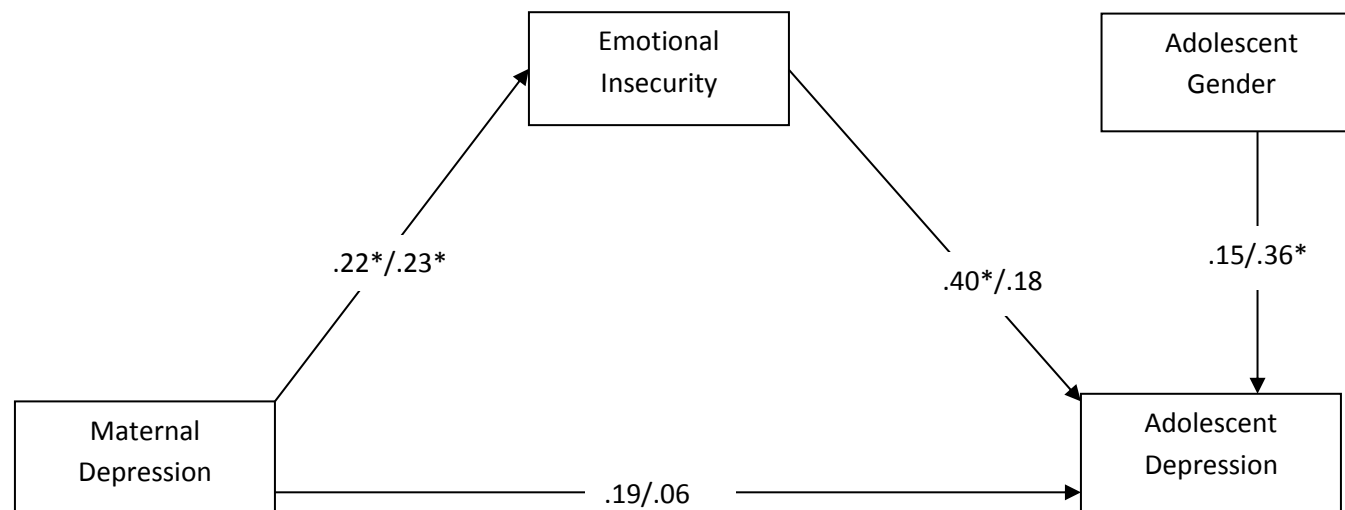


Figure 5. This model demonstrates the multiple group analysis of individuals with higher baseline systolic blood pressure (represented by first number) and individuals with lower baseline systolic blood pressure (represented by second number) when groups are allowed to differ. Error variances on dependent variables are estimated but not represented here. * indicates pathway is significant at $p < .05$. $N = 93$ participants

Figure 6. Cortisol as a Moderator

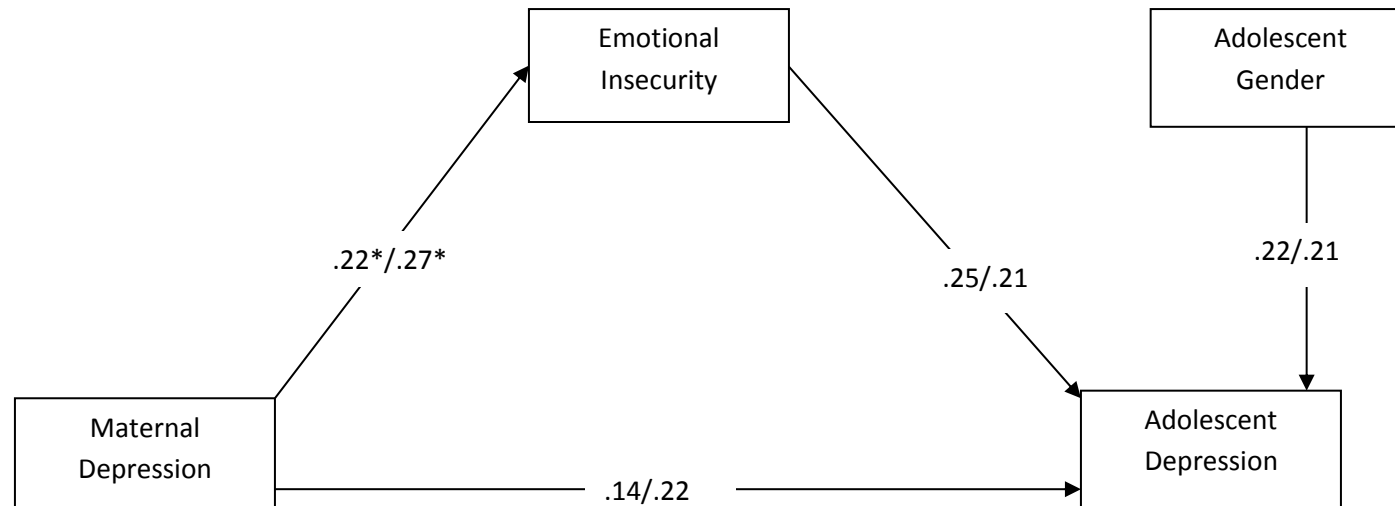


Figure 6. This model demonstrates the multiple group analysis of individuals with higher baseline cortisol levels (represented by first number) and individuals with lower baseline cortisol data (represented by second number) when groups are allowed to differ. Error variances on dependent variables are estimated but not represented here. * indicates pathway is significant at $p < .05$. $N = 80$ participants

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